

On pages 1-2, replace paragraph beginning on page 1, line 19, and ending on page 2, line 4, with the following amended paragraph.

The invention features a method for diagnosing a malignant neoplasm in a mammal by contacting a bodily fluid from the mammal with an antibody which binds to an human aspartyl (asparaginyl) beta-hydroxylase (HAAH) polypeptide under conditions sufficient to form an antigen-antibody complex and detecting the antigen-antibody complex (for the purposes of this specification, HAAH polypeptide refers to the amino acid sequence of SEQ ID NO:2 and HAAH cDNA refers to the nucleotide sequence of SEQ ID NO:3). Malignant neoplasms detected in this manner include those derived from endodermal tissue, e.g., colon cancer, breast cancer, pancreatic cancer, liver cancer, and cancer of the bile ducts. Neoplasms of the central nervous system (CNS) such as primary malignant CNS neoplasms of both neuronal and glial cell origin and metastatic CNS neoplasms are also detected. Patient derived tissue samples, e.g., biopsies of solid tumors, as well as bodily fluids such as a CNS-derived bodily fluid, blood, serum, urine, saliva, sputum, lung effusion, and ascites fluid, are contacted with an HAAH-specific antibody.

On pages 20, lines 21-27, replace the paragraph the following amended paragraph.

Under the terms of the Budapest Treaty on the International Recognition of the Deposit of Microorganisms for the Purpose of Patent Procedure, hybridoma FB501 (which produces monoclonal antibody FB50; designated ATCC accession no. PTA 3386), hybridoma HA386A (which produces monoclonal antibody 86A; designated ATCC accession no. 3385), hybridoma

HA15C7A (which produces monoclonal antibody 5C7; designated ATCC accession no. 3383), and hybridoma HA219B (which produces monoclonal antibody 19B; designated ATCC accession no. 3384) were deposited on May 17, 2001, with the American Type Culture Collection (ATCC) of 10801 University Boulevard, Manassas, Va. 20110-2209 USA.

On page 56, replace Table 4 with the following amended Table.

Table 4: Overexpression of enzymatically active HAAH indicates malignancy

Cdna	# of foci ± S.D. <sup>b</sup>	NIH 3T3 clone	# of colonies <sup>c</sup>
pcDNA3 (mock)	6.0 ± 3.3	pcDNA (mock)	0.4 ± 0.5
murine [H]AAH	14.0 ± 2.9	clone 18 <sup>d</sup>	6.2 ± 2.9
mutant murine [H]AAH <sup>a</sup>	1.6 ± 1.0	clone 16 <sup>e</sup>	4.7 ± 6.5
[human] HAAH	32.0 ± 5.4		
v-scr	98.0 ± 7.1		

a. enzymatically inactive [H]AAH

b. P<0.01 compared to mock and mutant murine [H]AAH

c. P<0.001 compared to mock

d. Clone 18 is a stable cloned NIH 3T3 cell line that overexpression human HAAH by approximately two fold.

e. Clone 16 is a stable cloned NIH 3T3 cell line that overexpresses human HAAH by about 50%.